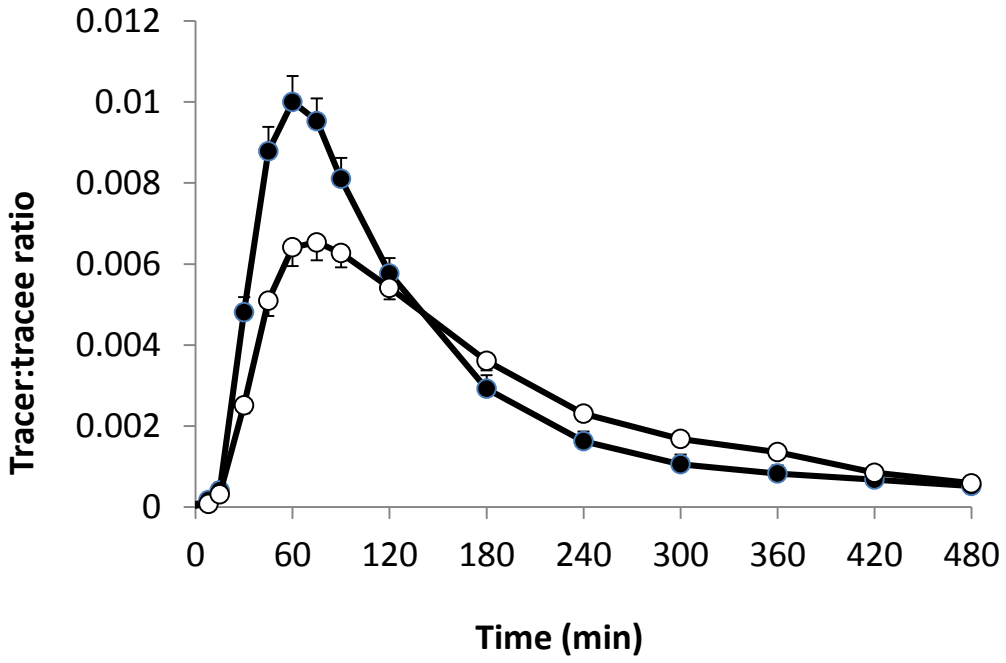

Figure S1: Mean tracer: trace ratio for glycerol stable isotope enrichment used to model VLDL-TG kinetics (n=58, mean ± SEM, filled circles represent $VLDL_1$-TG, open circles represent $VLDL_2$-TG)

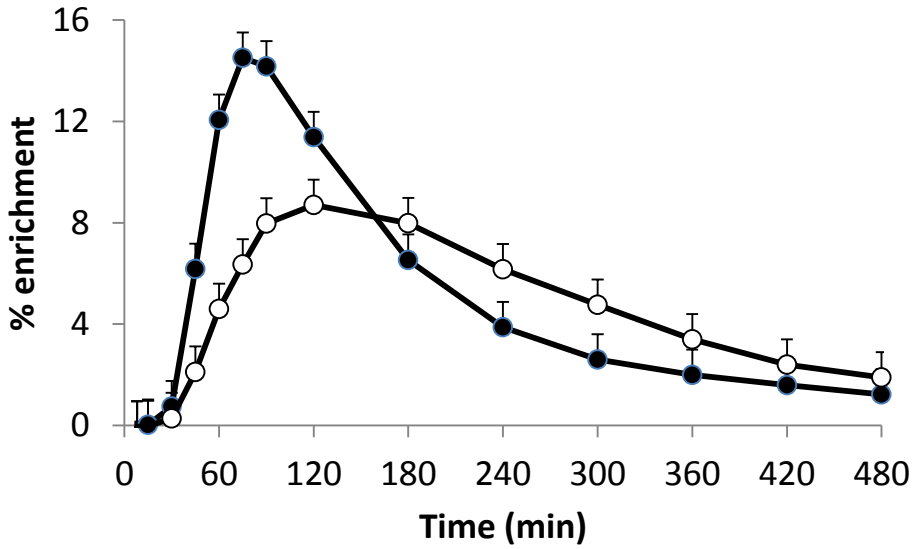

Figure S2: Mean percent enrichment for leucine stable isotope enrichment used to model VLDL-apoB100 kinetics (n=59, mean ± SEM, filled circles represent $VLDL_1$-TG, open circles represent $VLDL_2$-TG)

Figure S3

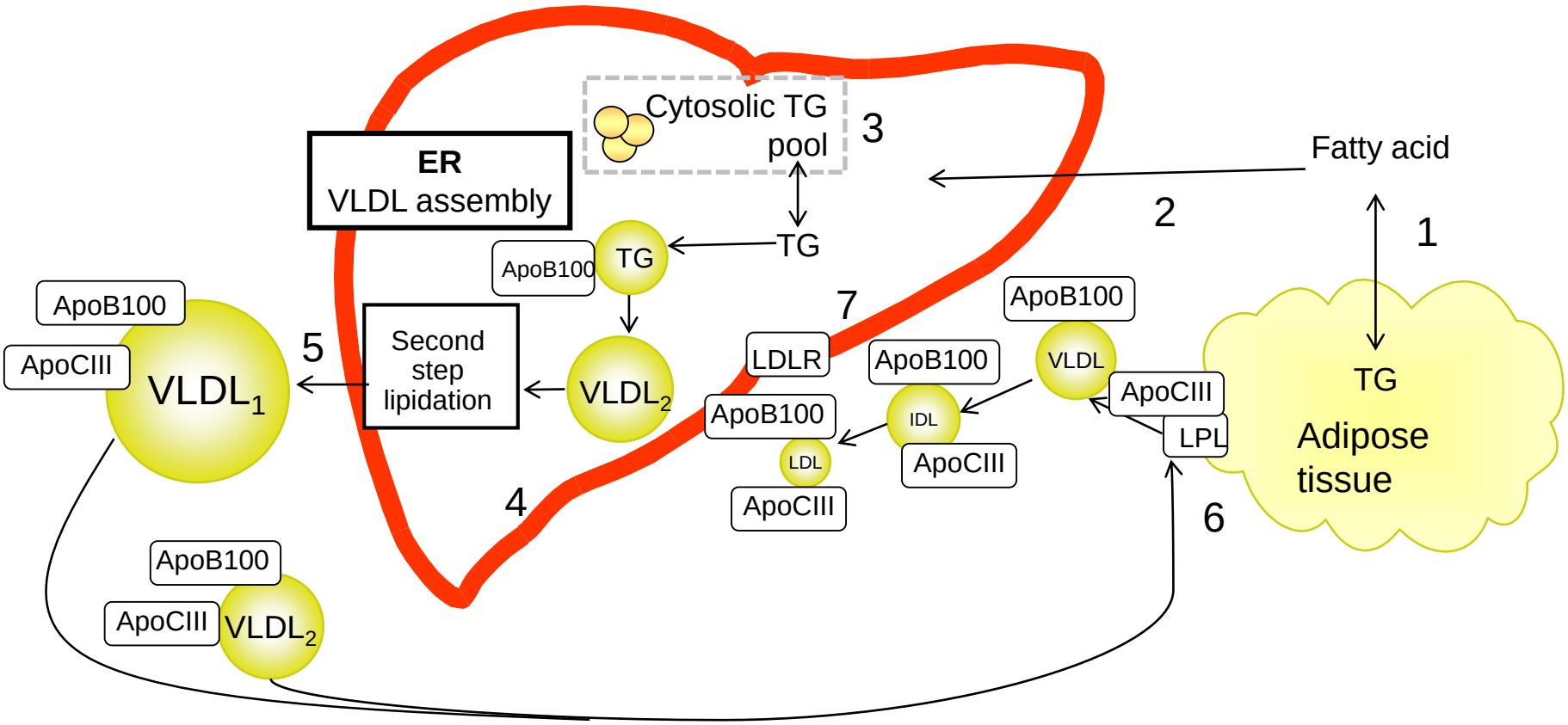
Cytosolic TG pool
3
ER
VLDL assembly
Fatty acid
2
1
TG
ApoB100
TG
ApoB100
ApoCIII
VLDL1
5
Second step lipidation
VLDL2
7
LDLR
ApoB100
VLDL
ApoB100
IDL
ApoCIII
LPL
ApoB100
LDL
ApoCIII
TG
Adipose tissue
4
6
ApoB100
ApoCIII
VLDL2

Figure S3: Overview of hepatic fatty acid trafficking according to abdominal obesity and menopausal status in healthy women.

1. Fatty acids are liberated from the intracellular lipolysis of adipose tissue triacylglycerol (TG) and the rate of appearance ((Ra) μmol.min^{-1} per kg fat mass) is significantly lower (P=0.01) with abdominal obesity. 2. Fatty acids in the systemic circulation taken up by peripheral tissues is described by a lower rate of disappearance ((R_d) μmol.min^{-1} per kg lean mass) in post-compared to pre-menopausal women (P=0.015). Fatty acids enter the liver and can be esterified to make TG which can remain (stored) in the liver. 3. Liver fat accumulation is higher with abdominal obesity (P<0.001). 4. TG can also be secreted in very low-density lipoprotein (VLDL) which contain one apoB100 per particle. $VLDL_2$-TG direct production (mg/kg lean mass) is higher in post- compared to pre-menopausal women (P=0.002). The effect of abdominal obesity on $VLDL_2$-apoB production (per day) is different in pre- and post-menopausal women (P<0.05). $VLDL_2$ particles are larger (more TG enriched) in post- compared with pre-menopausal women (P<0.05). 5. Rather than being directly secreted, $VLDL_2$ can be further lipidated to make TG rich VLDL particles which are known as $VLDL_1$. With abdominal obesity there is higher $VLDL_1$-TG production (mg/kg lean mass) (P=0.001). $VLDL_1$-apoB production is higher with abdominal obesity (P=0.001) and there was an interaction between abdominal obesity and menopause status (P<0.05). 6. VLDL particles in the systemic circulation are hydrolysed by lipoprotein lipase (LPL) which is located in many tissues, notably adipose tissue and skeletal muscle, producing intermediate density lipoprotein IDL. Higher plasma apoC-III:LpB concentrations after the menopause may play an important role by impairing LPL-mediated VLDL-TG hydrolysis and non-HDL (high density lipoprotein) cholesterol clearance (7) via the LDL receptor (LDLR)).